# THE CHANGE IN SYSTEMIC RESISTANCE BEFORE AND AFTER CORONARY ARTERY BYPASS GRAFTING AND ITS EFFECTS ON CARDIAC INDEX

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*This study investigated the effects of changes in systemic vascular resistance (SVR) on cardiac performance in patients undergoing CABG along with appropriate hemodynamic monitoring; and a mathematical limit value was explored that could guide the treatment for SVR. Patients were categorized into two groups on the basis of a SVR value greater than 1200 dyne/s/cm2 or lower than 1000 dyne/s/cm5. The hemodynamic parameters (heart rate, mean arterial pressure, pulmonary capillary wedge pressure, cardiac index, SVR) were compared between the two groups. When the parameters affecting the cardiac index (CI) were constant, the CI was low (2.1 ± 0.3 L/min/m2) only in cases with the higher SVR values (1591 ± 259 dyne/s/cm5), and the CI value was shown to increase (2.7 ± 0.2 L/min./m2, p < 0.001) upon the marked decline in SVR (832 ± 108 dyne/s/cm5) following the administration of nitroglycerine. It was concluded that nitroglycerine was necessary for lowering the SVR after CABG, and that the maintenance of a SVR value below 1000 dyne/s/cm5 was beneficial for cardiac hemodynamics.*

Coronary artery bypass grafting (CABG) technique was first introduced in 1967 for the prevention of angina pectoris, and since that time the mortality rates decreased from approximately 20% to 1% (1,2). Improved perioperative myocardial protection, technical advances, close hemodynamic monitoring during the post-operative period, and better knowledge of cardiac hemodynamics are the likely explanations for the decline in mortality rates (3,4,5). A more comprehensive knowledge of hemodynamics allows for precise and timely medical interventions, which serve to maintain cardiac performance (6,7). While the heart rate, ECG and central venous pressure were the only means of hemodynamic monitoring during early years of CABG technique, a wide range of hemodynamic parameters can be monitored during the post-operative period nowadays. Particularly with the introduction of Swan-Ganz catheter in 1970, complete monitoring of cardiac performance became possible, and by means of this new opportunity, a new era in hemodynamic monitoring commenced with the recognition of the fact that the filling pressure in the right heart did not always reflect the filling pressure in the left heart (8,9,10,11,12). Consequently, it was established that the rise in systemic vascular resistance (SVR, or afterload) following CABG causes an increase in the myocardial oxygen demand, compromising the ventricular myocardium (5,13,14,15,16,17).

In this study we investigated the effects of the change in SVR on cardiac performance in patients undergoing CABG along with appropriate hemodynamic monitoring with the aim of finding out a mathematical limit value that could guide the treatment decisions regarding SVR.

## MATERIALS AND METHODS

The study population consisted of 30 patients undergoing CABG and the patients were separated into two groups on the basis of post-grafting SVR values:

*Group I:* 16 male patients with a SVR value greater than 1200 dyne/s/cm5 (Mean age:56±9 y, age range:35-78 y)

*Group II:* 14 male patients with a SVR value less than 1000 dyne/s/cm5 (Mean age:56±9 y, age range: 40-75 y)

Patients with a SVR value between 1000 and 1200 dyne/s/cm5 were excluded.

The percentage of patients who suffered a myocardial infarction (MI) before CABG was 56% and 50% in the first and the second groups, respectively. The risk factors, angiography findings and the results of ventricular assessments are shown in Table I-III.

**Table I:** The Risk Factors in Patient Groups

| Risk Factors | Group I (n=16) | Group II (n=14) |
|---|---|---|
| Cigarette smoking | 13 | 13 |
| Diabetes Mellitus | 2 | 3 |
| Heredity | 11 | 9 |
| Hypertension | 8 | 7 |
| Hypercholesterolemia | 9 | 7 |
| Stress | 12 | 10 |
| Obesity | 0 | 1 |

**Table II:** The Results of the Coronary Angiography Assessments

| Angiography Results | Group I (n=16) | Group II (n=14) |
|---|---|---|
| Single vessel | 4 | 3 |
| Two vessels | 9 | 7 |
| Three vessels | 3 | 4 |
| Left Main Coronary Artery | 0 | 0 |

**Table III:** The Ventriculography Assessments

| Ventriculography | Group I (n=16) | Group II (n=14) |
|---|---|---|
| Normal | 8 | 8 |
| Moderately abnormal | 8 | 6 |
| Severely abnormal | 0 | 0 |
| Ventricular aneurysm | 0 | 0 |

Following premedication with diazepam (i.v., 10 mg) and ceftriaxone (i.v., 2 g) 1 hour ahead of operation, the heart rate and rhythm was monitored in all patients. A 16 G catheter was inserted into the right cephalic vein, a 18 G percutaneous catheter was inserted into the left radial artery. Induction and intubation in anesthesia were performed with 0.3mg/kg etomidate, 2mg/kg fentanyl, 0.1mg/kg pancuronium. For maintenance; 1-3 mg/kg/min fentanyl and 0.5-1 mg/kg/ hour midazolam were used. A 7 F Swan-Gantz (American Edwards), thermodilution catheter was inserted into the pulmonary artery via right internal jugular vein were placed in each patient to allow for continuous monitoring of right atrial, pulmonary artery and pulmonary capillary wedge pressures (PCWP). Cardiac performance (Cardiac Output: CO) was measured on the basis of thermodilution principle with an American Edwards COM-1

CO monitor, along with the administration of 5 cc of cold (0-5 °C) saline. Appropriate surgical procedures were commenced for CABG. Gambro PMO 10-220 roller pump and Bentley BCM 7 membrane oxygenator were used for CABG and the mean pump flow was 2 lt/min/m2 at the commencement of CPB. Bıcakcilar heater/cooler was used as the heat exchanger. Pump flow was lowered to 1.6 lt/dk/m2 while the patient was cooled to 28-30 C_. Following the placement of distal and proximal anastomoses, hemodynamic monitoring was continued in the intensive care unit, and 0.5 µg/kg/min nitroglycerine was given after the termination of CABG.

Hemodynamic assessments (heart rate, mean systemic arterial pressure, right atrial pressure, mean pulmonary artery pressure, PCWP) were made before the induction of anesthesia, at the termination of CABG, and at 1, 6, 12, and 18 h postoperatively. Cardiac index (CI) and SVR were estimated using the CO device and computer software.

Statistical assessments were performed with Microstat software, using the significance of the difference between mean values or chi-square tests. A p value less than 0.05 was considered significant, and a p value less than 0.001 was considered highly significant.

## RESULTS

The two groups were similar with respect to preoperative characteristics (age, preoperative symptoms, MI, risk factors, coronary angiography and ventriculography findings) and hemodynamic parameters ($p > 0.05$). The CI was low in the group with a high SVR ($p < 0.001$), and there were no significant differences between assessments in Group II.

The comparison of operative data between the groups is shown in Table IV, and no significant differences were detected with respect to these parameters ($p > 0.05$).

**Table IV:** The Comparative Operative Data Between the two Groups

| Angiography Findings | Group I (n=16) | Group II (n=14) | p |
|---|---|---|---|
| ACCT | 48.3±21.9 | 53.2±19.5 | NS |
| TGT | 93.0±32.4 | 106.9±38.2 | NS |
| NA | 2.0±0.7 | 2.2±0.8 | NS |
| NI | 3 | 2 | NS |
| IABP | 2 | 1 | NS |

**Abbreviations: ACCT, aortic cross-clamp time; TGT, total grafting time; NA, number of anastomoses; NI, need for inotropic agents; IABP; intra-aortic balloon pump; NS, not significant.**

These results demonstrate that both groups were similar with regard to preoperative and operative characteristics. Hemodynamic data were compared before CABG, at the termination of CABG, and at 1, 6, 12 and 18 postoperative hours, and the results are shown in Tables V-X.

**Table V:** The Hemodynamic Parameters Before CABG in Patient Groups

| | Group I (n=16) | Group II (n=14) | p |
|---|---|---|---|
| Heart rate (bpm) | 77.9±6.3 | 77.1±7.0 | NS |
| SMAP | 87.1±13.8 | 83.4±11.3 | NS |
| CVP | 6.2±2.3 | 5.4±2.2 | NS |
| PAMP | 14.1±1.9 | 14.2±1.6 | NS |
| PCWP | 10.1±1.7 | 9.4±1.9 | NS |
| CI | 2.3±0.4 | 2.5±0.4 | NS |
| SVR | 1274±187 | 1280±156 | NS |

**Abbreviations: SMAP, systemic mean arterial pressure (mmHg); CVP, central venous pressure (cmH2O); MPAP, mean pulmonary artery pressure (mmHg); PCWP, pulmonary capillary wedge pressure (mmHg); CI, cardiac index (Lt/min/m2); SVR, systemic vascular resistance (dyne/s/cm5)**

**Table VI:** The Hemodynamic Parameters After CABG in Patient Groups

| | Group I (n=16) | Group II (n=14) | p |
|---|---|---|---|
| Heart rate (bpm) | 83.3±11.4 | 81.9±11.5 | NS |
| SMAP | 75.7±10.5 | 76.2±9.4 | NS |
| CVP | 6.0±2.5 | 5.4±2.2 | NS |
| PAMP | 11.0±2.8 | 10.2±2.4 | NS |
| PCWP | 8.3±2.1 | 8.0±1.8 | NS |
| CI | 2.1±0.3 | 2.4±0.3 | < 0.001 |
| SVR | 1591±259 | 850±105 | < 0.001 |

**Abbreviations: please refer to Table V.**

**Table VII:** The Hemodynamic Parameters 1 Hour After CABG in Patient Groups

| | Group I (n=16) | Group II (n=14) | p |
|---|---|---|---|
| Heart rate (bpm) | 84.3±9.7 | 82.4±8.6 | NS |
| SMAP | 78.6±9.2 | 78.0±8.1 | NS |
| CVP | 6.1±2.0 | 5.6±1.9 | NS |
| PAMP | 10.6±2.4 | 9.5±2.1 | NS |
| PCWP | 8.2±1.2 | 7.5±1.8 | NS |
| CI | 2.2±0.2 | 2.5±0.3 | < 0.001 |
| SVR | 1581±252 | 853±81 | < 0.001 |

**Abbreviations: please refer to Table V.**

**Table VIII:** The Hemodynamic Parameters 1 Hour After CABG in Patient Groups

| | Group I (n=16) | Group II (n=14) | p |
|---|---|---|---|
| Heart rate (bpm) | 81.0±6.5 | 81.0±8.1 | NS |
| SMAP | 78.9±6.1 | 79.0±6.4 | NS |
| CVP | 3.1±1.4 | 3.7±0.8 | NS |
| PAMP | 9.2±2.2 | 9.6±2.6 | NS |
| PCWP | 6.5±1.0 | 6.3±1.0 | NS |
| CI | 2.8±0.1 | 2.7±0.1 | NS |
| SVR | 832±108 | 836±85 | NS |

**Abbreviations: please refer to Table V.**

**Table IX:** The Hemodynamic Parameters 12 Hours After CABG in Patient Groups

| | Group I (n=14) | Group II (n=16) | p |
|---|---|---|---|
| Heart rate (bpm) | 81.0±6.5 | 81.0±8.1 | NS |
| SMAP | 78.9±6.1 | 79.0±6.4 | NS |
| CVP | 3.1±1.4 | 3.7±0.8 | NS |
| PAMP | 9.2±2.2 | 9.6±2.6 | NS |
| PCWP | 6.5±1.0 | 6.3±1.0 | NS |
| CI | 2.8±0.1 | 2.7±0.1 | NS |
| SVR | 832±108 | 836±85 | NS |

**Abbreviations: please refer to Table V.**

**Table X:** The Hemodynamic Parameters 18 Hours After CABG in Patient Groups

| | Group I (n=16) | Group II (n=14) | p |
|---|---|---|---|
| Heart rate (bpm) | 79.5±4.5 | 80.8±5.1 | NS |
| SMAP | 78.3±5.5 | 82.0±6.8 | NS |
| CVP | 3.5±1.0 | 3.7±1.0 | NS |
| PAMP | 9.5±2.3 | 8.7±2.2 | NS |
| PCWP | 6.6±1.2 | 5.8±1.4 | NS |
| CI | 2.7±0.2 | 2.7±0.1 | NS |
| SVR | 822±87 | 855±73 | NS |

**Abbreviations: please refer to Table V.**

In Group I, SVR remained high until postoperative 12th with an associated low value for CI, and only after that point of time an increase was observed. The relationship between CI and SVR is shown in Figure 1, which demonstrates that the increase in CI in Group I was accompanied with a decrease in SVR.

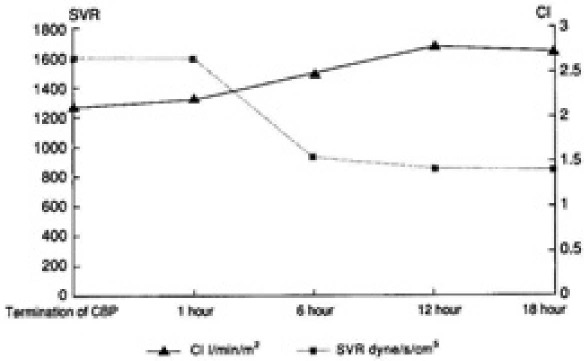


**Fig 1:** The relationship between SVR and CI

## DISCUSSION

Systemic arterial pressure is not only a function of the cardiac performance (CO), but also reflects the resistance or impedance produced by smaller arteries in the circulation (18,19). Maintenance of cardiac filling pressure at an optimal level is one of the most critical elements of open cardiac surgery, and this is particularly important for patients undergoing coronary artery surgery (7). The association between the afterload and preload has an influence on CO (9,21,22). SVR, aortic impedance, arterial wall resistance, intra-aortic blood column mass, and the viscosity of the blood are important determinants of left ventricular afterload (20); these factors also affect the myocardial contractility as well as the cardiac performance. If a normal heart is pushed to the limits of the preload reserve, the increase in afterload restrains the ventricular wall motions resulting in a decreased stroke volume in the left ventricle, thus an inverse statistical relationship between the stroke volume and peripheral resistance is observed (7,23). Although it is known that patients have a diminished ventricular reserve immediately following the termination of CABG, in this study the CI value was low in patients with a high SVR following CABG, and an increase occurred in CI together with a decrease in SVR (5,15,16,17); in addition, a significant increase in CO and left ventricular ejection fraction was observed without a change in mean arterial pressure, along with a decrease in vascular impedance caused by the diminished afterload (24,25,26). Thus, the nitroglycerine infusion was deemed to be sufficient for the desired hemodynamic effects and for decreasing the SVR, without the need for additional specific arterial vasodilators (27,28,29,30,31). In our study, there were no significant differences with respect to heart rate and the mean arterial pressures before and after the vasodilator therapy in patients who received pre- and post-operative vasodilator therapy. However, the vasodilator therapy resulted in a decrease in SRI and an increase in CI (from 2.2 ± 0.2 L/min/m2 to 2.7 ± 0.7 L/min/m2). Furthermore, along with a decrease in SVR at the postoperative 6th caused by postoperative nitroglycerine infusion, an increase in CI was observed ($p < 0.001$), and the CI values became similar in both groups when the SVR values between the groups became also similar.

Although a SVR value below 1200 dyne/s/cm5 was proposed previously, we found that a SVR value below 1000 dyne/s/cm5 was beneficial hemodynamically, and that nitroglycerine infusion by itself was sufficient for reducing SVR (15).

## REFERENCES


1. Flemma R.J., Mullen D.C., Lepley D., Assa J.: A comperative synchronous coronary surgery survival study. Ann Thorac Surg 28:547-553, 1987
2. Loop F.D., Lytle B.W., Cosgrove D.M.: Influence of internal mammary artery graft on 10 year survival and other cardiac events. N Eng J Med 314:1-9, 1986
3. Estefanous F.G., Tarazi R.C.: Systemic arterial hypertension associated with cardiac surgery. Am J Cardiol 46: 685-694, 1980
4. Kreulen T.H., Bove A.A., McDonough M.T., Sands M.T., Spann J.F.: The evaluation of left ventricular function in man. A comparison of methods. Circulation 51:677-688
5. Peng C.F., Kane J.J., Jones E.M., Murphy M.L., Straub K.D., Doherty J.E.: The adverse effect of systemic hypertension following myocardial reperfusion. J Surgical Res 34: 59-67, 1983
6. Hurst J.W.: The Heart, Arteries and Veins. 7th edition, New York, Mc Graw-Hill Inc, 1990, 35-71
7. MacGregor D.C., Covell J.W., Mahler G., Dilley R.B., Ross J.Jr: Relations between afterload, stroke volume, and descending limb of Starling's curve. Am J Physiol 227: 884-887, 1974
8. Ganz W., Donoso R., Marcus H.S., Forrester J.S., Swan H.J.C.: A new technique for measurements of cardiac output by thermodilution in man. Am J Cardiol 27:392-398, 1971
9. Sagawa K.: The end-sysolic pressure-volume relation of the ventricle: Definition, modifications and clinical use. Circulation 63: 1223-1227, 1981
10. Steele P., Davies H.: The Swan-Ganz catheter in the cardiac laboratory. Br Heart J 35: 647-652, 1973
11. Swan H.J.C., Ganz W., Forrester J.: Catheterisation of the heart in man with the use of flow-directed balloon-tipped catheters. N Engl J Med 283: 447-451, 1970
12. Weisel R.D., Berger R.L., Hechtman H.B.:

Measurements of cardiac output by thermodilution. N Engl J Med 292: 682-687, 1975
**13.** Estefanous F.G., Tarazi R.C., Viljoen J.F., El Tawil M.Y.: Systemic Hypertension following myocardial revascularisation. Am Heart J 85:732-738, 1973
**14.** Estefanous F.G., Tarazi R.C.: Systemic arterial hypertension associated with cardiac surgery. Am J Cardiol 46:685-694, 1980
**15.** Gordon R.J., Tavin M., Rawitscher R.E., Daicoff G.R.: Changes in arterial pressure, viscosity and resistance during cardiopulmonary bypass. J Thorac Cardiovasc Surg 69:552-561, 1975
**16.** Reduto L.A., Lewrie G.M., Reid J.W., Whissenand H.H., Noon G.P., Kanon D., Debakey M.E., Miller R.R.: Sequential postoperative assesment of left ventricular performance with gated cardiac blood pool imaging following aortocoronary bypass surgery. Am Heart J 101:59-66, 1981
**17.** Sladen R.N., Rosenthal M.H.: Spesific afterload reduction with parenteral hydralazine following cardiac surgery. J Thorac Cardiovasc Surg 78:195-202, 1979
**18.** Nichols W.W., Pepine C.J.: Left ventricular afterload and aortic input impedance: Implications of pulsatile blood flow. Prog Cardiovasc Dis 24:293-299, 1982
**19.** Soderstram C.A., Wasserman D.H., Cowley R.A.: Arterial monitoring catheters. A prospective study of use and complications. Crit Care Med 9:203-208, 1981
**20.** Sonnenblick E.H.: Instantaneous force-velocity-length determinants in the contraction of heart muscle. Circ Res 16:441-444, 1965
**21.** Imperial E.S., Levy M.N., Zieske H.: Outflow resistance as an independent determinant of cardiac performance. Circ Res 9:1148-1155, 1965
**22.** Ross J.Jr: Cardiac function and myocardial contractility: A perspective. J Am Coll Cardiol 1:52-55, 1983
**23.** Ross J.Jr: Afterload mismatch and preload reserve: A conceptual framework for the analysis of ventricular function. Prog Cardiovasc Dis 18: 255-258, 1976
**24.** Chatterjee K., Parmley W.W.: Vasodilator therapy for acute myocardial infarction and chronic congestive heart failure. J Am Coll Cardiol 1:133-136, 1983
**25.** Shine K.I., Kuhn M., Young L.S.: Aspects of the management of shock. Ann Intern Med 93: 723-727, 1980
**26.** Zelis R., Mason D.T., Braunwald E.: A comparison of the effects of vasodilator stimuli on peripheral resistance vessels in normal subjects and patients with congestive heart failure. J Clin Invest 47:960-970, 1968
**27.** Parmley W.W., Chatterjee K.: Vasodilator therapy. Curr Probl Cardiol 11:1-75, 1978
**28.** Stinson E.B., Holloway E.L., Derby G.C., Oyer P.E., Hollingsworth J., Greipp R.B., Harrison D.C.: Comperative hemodynamic responses to chlorpromazine, nitroprusside, nitroglycerin and trimetaphan immediately after open heart surgery. Circulation 51, 52: Suppl 1:26-33, 1975
**29.** Lappas D., Powell W.M.J., Dagett W.M.: Cardiac dysfunction in the perioperative period. Pathophysiology, diagnosis, treatment. Anesthesiology 47:117-137, 1977
**30.** Stinson E.B., Holloway E.L., Derby G.C., Copeland J.G., Oyer P.E., Buehler D.L., Greipp R.B.: Control of myocardial performance early after open-heart operations by vasodilator treatment. J Thorac Cardiovasc Surg 73:523-530, 1977
**31.** Tatar H., Oztürk O, Aslan M, Genç C., Balkanlı K, Gürler C: Koroner arter cerrahisinde nitrogliserin ve sodyum nitroprussidin hemodinamik etkilerinin karşılaştırılması. Türk Kardiyoloji Derneği Arşivi 17:156-160, 1989